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THE COLLOIDAL GOLD TEST ON SPINAL FLUID; IN PARESIS AND OTHER MENTAL DISEASES.*

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The goldsol (colloidal gold) test has appeared at a time most opportune for the laboratory diagnosis of paresis. Many workers have found that occasionally one or two of the other laboratory findings, namely, pleocytosis, globulin excess, positive serum and spinal fluid Wassermann reaction, may be negative repeatedly in a very apparent case of paresis. Although such a result is unusual, and can often be corrected by repeated spaced examinations, nevertheless, the few unproved cases are sources of annoyance for the laboratory worker and tend to discredit the value of the results in the opinion of the clinician, who has been led to believe that all the above named findings should constantly be found strongly positive in this disease. Our work was started in February, 1914, after the reported discovery that paresis and other luetic nervous conditions apparently give specific reactions to the colloidal gold test.

Zsigmondy, in 1901, laid the foundation for the methods which in 1912 were applied to the examination of spinal fluid by Carl Lange (1). Solutions of colloids are known to be electrically charged. An oppositely charged electrolyte or colloid will precipitate a colloid in solution, only in definite

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quantitative amounts. Zsigmondy used ten c. c. of a colloidal gold solution which was precipitated by one c. c. of a ten per cent. sodium chloride solution and found that different proteins would protect or inhibit precipitation of the gold solution, until a definite dilution containing a certain amount of the protein was reached. He was able to make quantitative estimations of pure proteins and determine whether a protein was pure. Lange undertook to use the method to establish the nature of the proteins precipitated from spinal fluid by ammonium sulphate solution. In applying the test to spinal fluid, however, he obtained, in conditions where an excess of proteins was present, a precipitation and not protection of the colloidal gold in solution, in the presence of an electrolyte (sodium chloride), when the latter was too weak a solution to precipitate the gold independently. He discovered that a reaction took place with certain dilutions of the spinal fluid, in syphilis of the nervous system and in paresis, and that this reaction did not occur in the nonluetic conditions. The nature of these different reactions has not been ascertained. Lange proposes the theory that they are evidences of varying qualitative mixtures of proteins.

Technic.—All glassware must be cleansed with acid, washed with distilled water, and dried by heat, or alcohol and ether. This we have adhered to, save that test tubes were thoroughly cleansed with tap water and completely dried with gauze but not sterilized—a procedure which has not given rise to a single flagrant error. All water for stock solutions or dilutions, must be double distilled over glass or silver, using corks which have previously been boiled for connections. Contact with rubber or other metals must be avoided. In a cleansed Jena glass beaker, 500 c. c. of double distilled water is heated gradually to 60° C., then five c. c. of one per cent. gold chloride and five c. c. of two per cent. potassium carbonate are added in rapid succession. The solution is heated quickly until the first

steam bubbles arise, when five c. c. of a one per cent., forty per cent. formaldehyde solution are added and the beaker is shaken until a red solution tinged with yellow is obtained. The final solution should be clear, transparent, and devoid of blue tints. The solution will keep many months. It is best to prepare new solutions of gold chloride, potassium carbonate, and formaldehyde solution each time the indicator is made up. Double distilled water, sterilized by boiling in a clean flask and a ten per cent. solution of sodium chloride C.P. complete the stock solutions. We keep these in the ice box.

To perform the test, eleven test tubes are placed in a row in a test tube rack. A 0.4 per cent. solution of sodium chloride is made from the ten per cent. stock solution. Into the first test tube 1.8 c. c. of a 0.4 per cent. sodium chloride are placed with a five c. c. pipette and one c. c. into each of the succeeding tubes. With a one c. c. pipette graduated to the tip, 0.2 c. c. of a spinal fluid is added to the first tube, making a one to ten dilution. After thorough mixing, one c. c. is transferred to the second test tube, giving a one to twenty dilution in the second tube. This is repeated until the tenth tube is reached, which will contain a one to 5,120 solution after the final c. c. of this mixture is discarded. The last test tube becomes a control. All tubes receive five c. c. Lange's colloidal gold solution and are shaken. The readings are taken the following day or after twelve to twenty-four hours, standing at room temperature. To indicate the reaction obtained, we have used the following nomenclature:

No. 0=red—original color.
No. 1=red blue.
No. 2=blue red.
No. 3=dark blue.
No. 4=pale blue.
No. 5=complete precipitation, colorless supernatant fluid.

In our opinion, little value can be placed on the

reaction before No. 3, or dark blue with absence of a red tinge, is reached. Since nearly all our fluids, obtained from insane patients, reacted in some tubes to the extent of No. 1 or No. 2, we must agree with Kaplan and McClellan (3), that a positive result must be confined to a reaction equal to or greater than No. 3 in psychiatric determinations.

Lange found that luetic fluids reacted in the first four or five tubes and gave the maximum of reaction in dilutions of one to forty, one to eighty and one to 160. A graphic representation produces a definite curve with the reaction in Lange's "luetic zone" (Table 1).

Lange and others have described a reaction found in acute meningitis and have termed it the "menin-

TABLE 1.—LUETIC CURVE OR ZONE.

Spinal Fluid dilution		1-10	20	40	80	160	320	640	1280	2560	5120
5	colorless										
4	pale blue										
3	dark blue										
2	blue red										
1	red blue										
0	red										

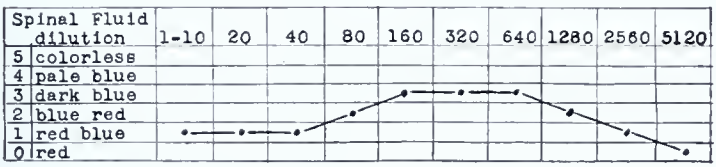
gitic zone" or curve. The greatest reaction is seen in the higher dilutions. (Table 2.)

A more specific curve has been described by Lange and others for general paralysis of the insane. A complete precipitation is usually obtained in the first four or five tubes. Miller and Levy (2) have termed this the "paretic zone." (Table 3.)

Results. Our work consists of 135 examinations on fluids from 111 patients. We undertook to examine the spinal fluid of all available paretics and patients afflicted with cerebrospinal syphilis in the State Hospital for Insane, Norristown, Pa. The clinical diagnoses are those prescribed by Dr. Jessie M. Peterson and Dr. S. Metz Miller, chief resident physicians of the female and male departments respectively. We have not eliminated obscure cases,

but have classified each in the grouping to which, after careful and prolonged consideration of the clinical aspects, each has been placed. We believe that by including all cases, our percentages of positives and results will more closely correspond with

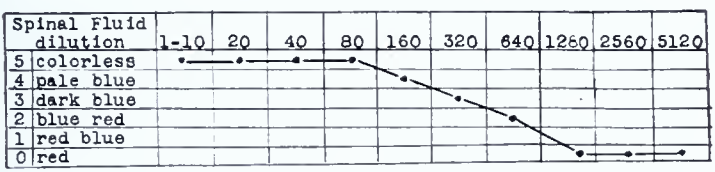
TABLE 2.—TYPE OF MENINGITIC CURVE.



that which the clinician may expect in support of his diagnoses.

The tabulated results require certain explanations. The blood serum Wassermann reaction is the strongest positive result obtained at any examination. The date indicates only the time of all spinal fluid examinations recorded. For the Wassermann reaction, the following technic is used: For the hemolytic system, we employ 0.5 c. c. of five per cent. solution washed sheep corpuscles, two units

TABLE 3.—PARETIC ZONE OR CURVE.



of high potency antisheep amboceptor, two units of complement (0.05 c. c.) each in total bulk of 0.5 c. c. Complement is titrated for each series of reactions. Patient's serum is used in 0.1 and 0.2 c. c. quantities, and spinal fluid in 0.2 and 0.4 c. c. quantities, properly controlled. No larger amount than 0.4 c. c. of spinal fluid has been used for any result recorded. As antigen, always a good alcoholic extract of beef heart has been employed, at times with other antigens. All spinal fluids and many bloods

have been examined in addition by the single unit system, namely, a single unit of corpuscles, amboceptor, and complement (0.05 c. c.) with 0.1 c. c. of serum or 0.2 c. c. spinal fluid. The result recorded is that provided by the double unit system, the weak positives of which are often made more apparent by the single unit system. The spinal fluid cell count was made by the Fuchs-Rosenthal chamber method. The globulin estimation was performed by the Noguchi butyric acid method and Ross method, using saturated ammonium sulphate solution.

Paresis. Spinal fluids from seventy patients were obtained and of this number, fifty-three gave complete reduction or a five plus reaction in the first tubes. Thus, 75.71 per cent. of the total number of paretics provided the characteristic complete reduction in the "paretic zone." Nine other cases, LIV to LXII inclusive, showed almost complete reduction of the gold or four plus reactions in the first tubes. We believe these should be included among those affording a definite positive reaction for paresis. There is but a shade of difference in the degree of the reaction, and it is possible almost to certainly render a positive laboratory diagnosis of paresis on this result in our experience. We would therefore judge our first sixty-two cases as definitely positive results and obtain thereby a percentage of 88.57. Four cases, LXIII, LXIV, LXV, LXVI, gave a single strong reaction in the first or second tube. These we would interpret as doubtful but probable cases of paresis from the laboratory standpoint in suspected cases and figure 5.71 per cent. of the whole number of paretics. If we add these to the positive cases, we might say that 94.28 per cent. of corroborative evidence of paresis was afforded to the clinical diagnosis of unselected cases by careful interpretation of the result. One could scarcely expect higher percentages in a group of paretics in all stages without selection. Kaplan (4) found a positive paretic curve in ninety-five per

cent. of his cases. Miller and Levy (2) report 100 per cent. of positive reactions. Three of our cases, LXVII, LXVIII, LXIX, clinically paresis or tabetoparesis, gave only the typical luetic curve with the strongest reaction, a three or four plus, in the third, fourth or fifth tube. Nothing specific for the diagnosis of paresis can be obtained from these results, which constitute 4.28 per cent. of the whole number. Lange, in his original work, found that his paretics gave a maximum reaction of 5, 4 or 3 plus. One case, 1.42 per cent, LXX, believed to be paresis of two years' duration, without physical signs of the disease, has given an absolutely negative result by the goldsol test on two examinations, also negative spinal fluid Wassermann reactions.

The most important finding in paresis, we think, is demonstrated by the result in cases which at times or repeatedly, have given a very weak positive or negative spinal fluid Wassermann reaction. Of this type are Cases VI, XIII, L, LII, LIV, LVII, LXVI, the first six of which gave typical paretic curves, the last a doubtful paretic reaction. Case LXVIII, with a weak cerebrospinal fluid Wassermann, a demented paretic or possibly cerebral syphilis, showed only the luetic curve, and Case LXX, that of a paretic without physical signs, gave negative goldsol and cerebrospinal Wassermann. Therefore seven of nine, seventy-seven per cent., or ten per cent. of the whole number of paretics were established by the goldsol test when the cerebrospinal Wassermann had practically failed, for we regard a one plus Wassermann result of a very doubtful diagnostic value and within the limit of possible experimental error. Practically all negative or weak positive Wassermann results, both blood and spinal, in paresis, tabes, and cerebrospinal syphilis, have been verified by at least one other examination, and the same may be said of weak goldsol reactions in these conditions, except as included in the tables. It might well be stated here that the marked pleocytosis and globulin excess in paresis is regarded only as evi-

TABLE 4.—PARESIS.

No.	Name.	Date.	Blood. W. R.	Cerebrospinal fluid—W. R.	Cells.	Globulin—Nog.	Ross.	Gold reaction 1	2	3	4	5	6	7	8	9	10	Remarks.
1	M. K.	2-11-14	++++	++++	81	++	++	5	5	5	5	5	4	3	1	0	0	Verified by autopsy.
2	A. L. K.	2-27-14	+	++++	66	++	++	3	5	**5**	4	4	3	2	1	0	0	
3	J. M.	4-10-14	++++	++++	92	++	++	5	5	5	5	5	4	3	2	1	1	
4	J. A.	4-10-14	++++	++++	32	++	++	5	5	5	5	4	4	3	2	0	0	
5	F. Y.	4-30-14	++++	+++	15	++	++	5	5	3	4	4	3	3	2	1	0	Demented, progressive.
6	E. G.	5-16-14		+	65	++	++	5	5	5	5	4	4	3	2	1	0	Demented, progressive.
	E. G.	9-20-12	++	++++	20	+	++											Verified by autopsy.
7	M. R.	5-16-14	++++	++++	42	++	++	5	5	**5**	4	4	4	3	3	2	1	
8	E. G. W.	5-16-14	++++	++++	130	++	++	4	5	5	4	4	3	3	2	1	0	Tabetoparesis.
9	G. W.	5-16-14	++	+++	120	++	++	5	5	5	5	5	4	4	3	2	1	Tabetoparesis.
10	W. H.	5-16-14	+++	++++	48	++	++	5	5	4	3	4	3	3	3	2	1	In remission.
11	J. S.	5-17-14	++++	++++	26	++	++	5	5	5	5	4	4	3	2	1	0	
12	C. F.	5-17-14	++++	++++	41	++	++	5	5	5	**5**	4	4	2	1	1	0	
13	G. L.	5-17-14	Neg.	+	88	++	++	**5**	5	5	5	4	4	2	1	1	0	Terminal dementia.
14	T. W. H.	5-17-14	++	++++	36	++	++	5	5	5	5	4	4	3	2	1	0	
15	N. P.	5-17-14	++++	++++	32	++	++	5	5	5	4	3	3	2	1	1	0	
16	T. S.	5-17-14	++++	++	48	++	++	5	5	5	4	4	4	3	2	1	0	Terminal stages.
17	J. D.	5-19-14	++++	++++	105	+	++	4	5	5	5	5	4	4	3	2	1	
18	H. F.	5-19-14	++++	+++	14	+	++	5	5	5	5	5	4	4	2	1	0	
19	M. R.	5-19-14	++++	++	150	++	++	5	5	5	5	5	4	4	3	3	2	Verified by autopsy.
20	C. H.	5-19-14	++++	++	155	+	+	5	5	5	**5**	5	4	4	2	2	1	Advanced dementia.
21	C. S.	5-19-14	+++	++++	125	++	++	5	5	5	5	3	4	4	3	2	1	
22	M. K.	5-25-14	+++	++++	20	++	++	5	5	5	5	4	4	3	2	1	0	Remission.
23	J. L.	5-25-14	++++	++++	20	+	+	5	4	4	3	3	3	2	1	0	0	
24	W. M.	5-25-14	++	++	61	++	++	5	5	4	4	4	3	2	1	0	0	
25	C. H.	5-25-14	++++	++++	80	++	++	5	5	5	4	4	3	2	2	1	0	
26	H. R.	5-26-14	++++	++	60	++	++	**5**	5	5	4	4	4	3	2	1	0	Tabetoparesis.
27	R. B.	5-26-14	++	++	28	++	++	5	5	5	4	3	3	2	2	1	0	Terminal stage.
28	E. L.	5-26-14	++++	++++	46	++	++	5	5	5	4	4	4	3	2	1	0	
29	F. M.	5-26-14	+++	++++	290	++	++	5	5	5	4	4	4	3	2	1	0	
30	J. W.	5-26-14	++++	++++	22	++	++	5	5	5	5	4	4	3	3	2	1	
31	H. W.	6- 1-14	±	++	22	++	++	5	5	4	4	3	2	2	1	0	0	Terminal stage.
32	H. V. F.	6- 1-14	+++	++++	37	++	++	5	5	4	3	3	2	1	0	0	0	
33	E. S.	6- 1-14	+++	++++	32	++	++	5	5	5	4	3	2	1	0	0	0	
34	W. McE.	6- 2-14	++	++++	110	++	++	5	5	5	5	5	4	3	2	1	0	
35	S. G.	6-30-14	+++	+++	16	+	+	5	5	5	4	4	2	3	1	0	0	
36	L. C. O.	7-14-14			30	+	+	5	5	5	4	4	3	2	2	1	0	

																		Terminal stage.
45	S. G.	9- 1-14	++	++++	26	++	++	5	5	5	5	5	4	3	2	1	0	
46	F. J. R.	9- 8-14	++++	++++	160	+	+	5	5	4	4	3	2	1	0	0	0	
47	H. C.	9-15-14	+	+++	28	+	+	5	5	5	4	3	2	1	0	0	0	
48	C. M.	9-15-14	++++	++++	56	++	++	5	5	5	5	4	3	2	2	1	0	Treat. salv. early paresis.
	C. M.	7-28-14	Neg.	±	20	±	+	2	2	3	3	1	0	0	0	0	0	
49	R. W. B.	9-15-14	+++	+++	93	++	++	5	5	4	4	3	3	2	1	0	0	
50	J. W.	2-11-14	++++	Neg.	8	Neg.	+	5	5	5	5	4	3	1	0	0	0	Advanced dementia.
51	J. R.	7-28-14	Neg.	++	27	+	+	5	5	4	3	3	1	0	0	0	0	
52	L. S.	5-17-14	+++	Neg.	22	++	++	5	5	5	5	5	4	3	3	2	1	In remission.
53	I. F.	10- 6-14	+++	++++	27	++	++	5	4	3	3	3	2	1	0	0	0	
54	G. M.	6- 1-14		+	28	±	+	4	2	2	3	1	0	0	0	0	0	Convulsions, advanced dementia.
	G. M.	9-15-14	++++	±	21	±	+	4	5	4	3	3	2	2	1	0	0	
55	H. S.	2-18-14	+++	+++	33	++	++	4	4	4	4	3	3	3	2	1	0	
56	H. H.	5-16-14	++++	+++	30	Neg.	±	4	4	4	4	3	3	2	1	0	0	In remission.
57	F. P.	2-27-14	++++	±	140	±	±	4	4	3	3	2	0	0	0	0	0	
58	C. P.	5-26-14	+++	++	76	+	++	4	4	3	3	3	3	2	1	0	0	Blind, advanced dementia.
59	C. A.	5-16-14	++++	++	10	++	++	4	4	3	3	3	3	2	2	1	0	Advanced dementia.
60	J. S.	6- 1-14	+++	++++	16	++	++	4	2	2	3	2	1	0	0	0	0	
61	H. W.	6- 2-14	++	++++	26	++	++	4	4	2	3	2	2	1	0	0	0	
62	W. K.	6- 9-14	+++	++++	57	++	++	4	4	3	3	3	2	1	0	0	0	
63	H. S.	5- 9-14	++++	+++	80	+	+	4	2	1	1	1	1	0	0	0	0	Tabetoparesis, in partial remission
63	H. S.	9-15-14		+++	160	+	+	2	2	3	3	2	1	0	0	0	0	
64	L. H.	6- 9-14	++	+++	72	±	+	4	2	3	3	2	1	0	0	0	0	Advanced dementia.
65	L. L.	6- 9-14	++++	++	27	+	+	1	2	2	2	1	1	0	0	0	0	Slow deterioration in remission.
65	L. L.	7-15-14		++	72	±	+	4	1	1	1	1	0	0	0	0	0	
66	D. B.	5-20-14	++++	±	24	±	+	2	4	3	3	3	2	2	1	1	0	Demented, in remission.
66	D. B.	6- 1-14		Neg.	88	±	±	3	2	2	1	0	0	0	0	0	0	
67	L. W.	5-19-14	++++	++++	130	±	—	1	2	4	4	3	3	2	1	0	0	Imbecile, early tabetoparesis.
68	F. S.	5-25-14	+++	Neg.	36	—	±	2	2	3	3	2	1	0	0	0	0	Demented paretic, possibly cerebral syphilis.
68	F. S.	9-22-14		+	9	±	±	1	2	2	2	1	0	0	0	0	0	
69	C. B.	6- 2-14	+++	+++	90	++	++	2	2	3	3	2	1	0	0	0	0	Juvenile in remission.
70	R. L.	6- 2-14	+++	Neg.	68	++	++	1	1	1	1	1	0	0	0	0	0	Two years' duration; no phys. signs of paresis.
70	R. L.	7-14-14		Neg.	8	+	+	2	2	2	2	1	1	0	0	0	0	

TABLE 5.—TABES.

No.	Name.	Date.	Blood. W. R.	Cerebrospinal fluid. W. R.	Cells.	Globulin. Nog.	Ross.	Gold reaction. 1	2	3	4	5	6	7	8	9	10	Remarks.
71	I. DeH.	4-10-14	—	—	8	—	±	3	3	4	4	4	3	2	0	0	0	Verified in autopsy.
72	F. H.	5-19-14	—	—	28	—	±	2	2	3	4	4	3	2	1	0	0	Dementia præcox and tabes.
73	D. S.	5-19-14	—	—	16	—	±	1	1	2	2	1	1	0	0	0	0	Dementia and tabes, stationary.
74	F. M.	6-16-14	++	+	95	±	+	2	2	2	1	1	1	0	0	0	0	Tabes, stationary.
74	F. M.	9-22-14		—	3	±	±	2	2	1	1	1	0	0	0	0	0	

TABLE 6.—CEREBROSPINAL SYPHILIS.

No.	Name.	Date.	Blood. W. R.	Cerebrospinal fluid. W. R.	Cells.	Globulin. Nog.	Ross.	Gold reaction. 1	2	3	4	5	6	7	8	9	10	Remarks.
75	E. C.	2-11-14	++++	+	39	—	±	1	1	2	3	3	2	1	0	0	0	
76	H. McG.	4-30-14	++++	+++	170	+	++	2	2	2	4	4	3	3	2	1	0	
77	M. S.	3-18-14	++++	—	16	—	+	2	3	3	3	3	2	1	0	0	0	
78	M. A. W.	3-11-14	++	±	31	+	±	1	2	3	3	3	1	0	0	0	0	
79	J. C.	5-16-14	++++	+	23	+	+	2	3	4	4	3	3	2	1	0	0	
80	M. E. W.	7- 8-14	++++	—	5	—	±	5	2	3	3	3	2	1	0	0	0	Slow pupillary light reaction, demented.
81	G. H.	5-17-14	+++	—	8	—	—	2	2	2	2	1	0	0	0	0	0	Irregular pupils, stationary.
82	M. L. T.	7- 8-14	++	—	34	—	+	2	2	2	1	1	0	0	0	0	0	
83	N. F.	8-18-14	+	+++	27	+	+	2	2	2	2	2	1	0	0	0	0	Hemiplegia.
84	M. S.	5-26-14	+++	—	16	±	+	1	1	2	2	1	0	0	0	0	0	Demented, stationary, physical signs.

No.	Name.	Date.	Blood. W. R.	Cerebrospinal fluid — W. R.	Cells.	Globulin — Nog.	Ross.	Gold reaction 1	2	3	4	5	6	7	8	9	10	Remarks.
85	E. K.	2-11-14	+++	—	6	—	—	1	1	1	1	1	0	0	0	0	0	Dementia præcox.
86	L. G.	6-15-14	++	—	11	+	+	2	2	2	1	1	1	0	0	0	0	Dementia præcox.
87	I. W.	7- 8-14	++++	—	4	—	±	1	1	1	1	1	0	0	0	0	0	Dementia præcox.
88	B. K.	6-15-14		—	120	±	+	2	2	2	1	1	0	0	0	0	0	Dementia præcox. No
88	B. K.	7-14-14	++++	—	60	+	+	4	3	3	3	2	1	0	0	0	0	evidence of nervous syphilis.
89	A. T.	9- 1-14	—	—	1	—	—	2	1	1	1	0	0	0	0	0	0	Dementia præcox.
90	A. S.	6- 9-14	—	—	2	—	±	1	1	0	0	0	0	0	0	0	0	Dementia præcox.
91	A. Y.	10- 6-14	++	—	7	—	—	1	1	1	0	0	0	0	0	0	0	Dementia præcox.
92	J. J.	10- 6-14	+	—	4	±	—	1	1	1	0	0	0	0	0	0	0	Dementia præcox.
93	A. Y.	10- 6-14	++++	—	3	—	—	1	1	1	0	0	0	0	0	0	0	Dementia præcox.
94	E. M.	3-30-14	—	—	0	—	±	1	1	1	1	0	0	0	0	0	0	Alcoholic psychosis.
95	C. N.	5-26-14	+++	—	12	±	+	2	2	2	2	2	0	0	0	0	0	Alcoholic psychosis.
96	W. D.	7-14-14	++++	—	12	±	+	2	2	1	1	0	0	0	0	0	0	Alcoholic psychosis.
97	E. B.	4-30-14	—	—	0	—	±	1	1	1	1	0	0	0	0	0	0	Manic depressive.
98	V. W.	5-20-14	—	—	0	—	±	1	1	2	2	2	2	1	0	0	0	Manic depressive.
99	J. J. F.	9- 8-14	—	—	20	—	—	1	1	0	0	0	0	0	0	0	0	Manic depressive.
100	D. B.	10- 6-14	+++	—	5	—	—	1	1	1	0	0	0	0	0	0	0	Manic depressive. (Thrombotic softening.)
101	T. T.	4-10-14	++	—	0	—	—	1	1	1	0	0	0	0	0	0	0	Gross brain lesion.
102	J. O.	6- 2-14	+++	—	10	±	+	1	1	1	1	0	0	0	0	0	0	Gross brain lesion.
103	H. L.	6- 9-14	++++	—	33	+	+	1	1	1	0	0	0	0	0	0	0	Gross brain lesion.
104	A. S.	9-29-14	—	—	17	++	++	2	2	2	2	2	2	1	0	0	0	Gross brain lesion.
105	E. G. H.	9-29-14	—	—	3	±	+	1	1	1	0	0	0	0	0	0	0	Gross brain lesion.
106	L. T.	4-30-14	++++	—	6	—	±	1	1	1	1	1	0	0	0	0	0	Senile psychosis.
107	H. G.	6-16-14	—	—	4	—	±	1	1	1	1	1	0	0	0	0	0	Senile psychosis.
108	J. McC.	6- 2-14	+++	—	32	±	±	1	1	1	0	0	0	0	0	0	0	Paranoia.
109	E. R.	10- 6-14	++++	—	2	—	—	1	1	1	0	0	0	0	0	0	0	Paranoia.
110	H. B.	5-19-14	—	—	3	—	±	1	1	2	2	1	0	0	0	0	0	Imbecility.
111	M. S.	2-11-14	—	—	3	—	—	1	1	1	1	0	0	0	0	0	0	Unclassified psychosis.

TABLE 8.—COMPOSITE TABLE OF RESULTS.

Disease.	No. of cases.	Blood Wass. Pos.	Blood Wass. Neg.	Spinal Wass. Pos.	Spinal Wass. Neg.	Ross test. Pos.	Ross test. Neg.	Noguchi test. Pos.	Noguchi test. Neg.	Pleocytosis.	Gold reaction Pos. paretic zone.	Gold reaction Doubt. paretic reaction.	Gold reaction Luetic zone.	Gold reaction Neg.
Paresis	79	66	4	65	5	66	4	63	7	69	62	4	3	1
Tabes	4	1	3	1	3	1	3	0	4	3			2	2
Cerebrospinal lues	10	10	0	4	6	6	4	4	6	8			6	4
Dementia præcox	9	7	2	0	9	2	7	2	7	2			1	8
Alcoholic psychosis	3	2	1	0	3	2	1	0	3	2				3
Manic depressive psychosis	4	1	3	0	4	0	4	0	4	1				4
Psychosis with brain lesion	5	3	2	0	5	4	1	2	3	2				5
Senile psychosis	2	1	1	0	2	0	2	0	2	0				2
Paranoia	2	2	0	0	2	0	2	0	2	1				2
Imbecility	1	0	1	0	1	0	1	0	1	0				1
Unclassified psychosis	1	0	1	0	1	0	1	0	1	0				1

dence of meningeal irritation, and while always expected in paresis, has no definite specific value in diagnosis. Again, other luetic nervous diseases produce a positive cerebrospinal Wassermann in a variable percentage of cases, but these conditions usually give the luetic curve with the goldsol test.

While this characteristic paretic curve will undoubtedly be of the greatest value in establishing or proving the diagnosis of paresis, we are inclined to believe that the reaction is dependent on the presence in the spinal fluid of certain proteins produced by parenchymatous degenerative nervous tissue changes, commonly due to syphilis, as in paresis or cerebral lues, but not necessarily of luetic origin. In short, the reaction is probably not specific for paresis, but the result of pathological changes, more often found in paresis than other neurological conditions and hence designated the paretic type, curve or zone. These views are supported by the findings of paretic curves by Miller and Levy (2) in one case of brain abscess, and two cases of epidemic meningitis, and by a similar finding in a case of multiple sclerosis by Kaplan and McClelland (3). It has not been demonstrated that certain other forms of meningoencephalitis do not give the paretic goldsol reaction in the spinal fluid. The result in trypanasomiasis, a protozoal type, may prove interesting.

Since it is more recently believed that tabes and cerebrospinal syphilis are but more localized and interstitial types of nervous lues, in contradistinction to paresis, a more diffuse and parenchymatous involvement, there appears to be no good reason why gradations of the latter process may not be found associated with tabes and cerebrospinal syphilis. This we know is so in tabetoparesis, in which the goldsol paretic curve is also found. It is but logical to expect that clinical cerebrospinal syphilis, if extensive, may produce a similar reaction, assumed by us to be dependent mostly on parenchymatous alterations. Such paretic curves in

cerebrospinal lues were occasionally obtained by Kaplan and McClelland (3) and by Miller and Levy (2) according to our interpretation and also by several German observers.

To certain other observations we desire to direct attention. In many of the weaker reactions, Cases XLVIII, LIV, LX, LXI, LXIV, LXVI, LXVII, LXVIII, a distinct luetic curve, or strong reaction in the third, fourth or fifth tube, is found, together with a stronger reaction in the first or second tube, or alone. It is possible that very early in paresis, only the luetic curve of localized, interstitial or vascular syphilis is present. This view is supported by Cases XLVIII and LXVII, both early cases. With the incidence of diffuse parenchymatous nervous syphilis, exemplified by paresis, certain additional proteins reaching the spinal fluid may produce the characteristic curve and reduction, which in most cases is maintained throughout the course of the disease. Cases LXIII, LXV, LXIX, seem to demonstrate that in remission, these proteins found in paresis may be partially or wholly lost, leaving only the luetic reaction. This may also be seen in Cases LXIV, LXVI, LXVIII, which are advanced paresis with dementia. It is in these stages that our experience has taught us to expect a weaker or variable cerebrospinal Wassermann, pleocytosis or globulin reaction. Intensive treatment probably will produce, if favorable, a reaction similar to that seen in these cases in remission, and we suggest that the goldsol test be employed to help control the results obtained by the use of intraspinous salvarsan or salvarsanized serum.

Findings in other psychoses. Many cases recorded among the nonluetic psychoses gave positive serum Wassermann reactions before the spinal fluid was examined. These have in most part advanced beyond the secondary stage of syphilis. Apart from these it will be noted that all our cases reacted to the extent of one or two plus in the first tube, probably a result due to the presence in the spinal fluid

of certain proteins common to all types of cerebral disease. These results early compelled us to place no confidence in a reaction which did not reach a maximum of three plus or dark blue without traces of red.

Accordingly, we obtained in tabes but two, LXXI, LXXII, of four cases, fifty per cent., providing a luetic curve of sufficient intensity to be termed positive. Cases LXXIII and LXXIV are old and stationary. Cerebrospinal syphilis furnished six positive luetic curves, LXXV to LXXX inclusive, in ten cases or sixty per cent. The value of the goldsol test in these two luetic diseases is shown by the fact that, of the total of eight positive results, five gave negative cerebrospinal Wassermann reactions and two doubtful weak positive reactions. On the other hand, one case of cerebrospinal syphilis, LXXXIII, gave a positive cerebrospinal Wassermann and a negative goldsol test. The luetic curve is in no way specific for these individual pathological conditions. Case LXXX gave complete reduction in the first tube, a condition very suggestive of paresis. Case LXXXVIII, under dementia præcox also furnished, on one examination, a paretic curve. The diagnosis, at first believed to be paresis, is now revised to dementia præcox. These two cases gave a very strong serum and a negative fluid Wassermann and their courses will be watched with interest.

Excluding this last named case of dementia præcox, we did not obtain a positive reaction in, nor a characteristic curve for any psychosis of nonluetic origin. This group is composed of twenty-seven cases. It is possible that these weak reactions may provide a rough index of the extent of organic brain disease in nonluetic psychoses, or, with the development of a more delicate technic, also furnish different types of curves for various psychoses.

CONCLUSIONS.

1. In our work with psychiatric cases, all reacted to the extent of one or two plus in the first tubes,

and hence no reaction less than three plus or dark blue was recorded as positive.

2. In paresis, the test is of great value in diagnosis, at least ninety per cent. providing the typical curve, the true specificity of which we doubt. The goldsol test should be used in conjunction with the serum and spinal fluid Wassermann reaction, globulin tests, and cell count, as a more delicate method of interpreting their results and to correct them when they fail.

3. Our cases of tabes furnished a marked luetic curve in fifty per cent.; cases of cerebrospinal syphilis in sixty per cent.

4. Nonluetic psychoses produce weak reactions, without characteristic curves, so far as we have ascertained.

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